patient no longer required blood transfusions. UIE increased to maximally 15.6 mg/24 h. A sharp decrease in ferritin level was observed (Fig. 1).

Increasing erythropoiesis during iron chelating therapy with DFO has been noticed in a number of studies. In patients with myelodysplastic syndrome as well as myelofibrosis, a reduction in blood transfusion needs was observed during treatment with DFO [2]. The response to recombinant erythropoietin, in patients with anemia of end stage renal failure and hemosiderosis, was improved by DFO. In patients with rheumatoid arthritis, a rise of Hb was observed during treatment with DFO and L1. Although aluminum chelating effects or reduction of disease activity cannot be excluded, ferrokinetic changes can explain the response [3].

After mobilisation of iron from the iron stores, not only iron excretion in urine is increased, but it may also be redistributed to the hematopoietic tissues by the chelator itself or by exchanging it with transferrin. In vitro studies have demonstrated that DFO can stimulate transferrin receptor expression on the erythroblast [4]. It has also been shown that iron chelators can pass the erythroblast membrane [5].

Before starting treatment with L1 our patient required about two to four URC each month. Repeated bone marrow biopsy showed no changes. We believe that iron kinetic changes due to L1 can possibly explain the observed phenomenon in our patient.

In view of these speculations, further research of the pharmacokinetics and effects of L1 and other iron chelators on iron metabolism and iron (re)distribution is necessary.

M.E.P. Smeets
G. Vreugdenhil
R.S.G. Holdrinet
Department of Hematology, University Hospital Nijmegen, the Netherlands and Department of Internal Medicine, St. Joseph Hospital, Veldhoven, the Netherlands

References

1. Kontoghiorges GJ, Bartlett AN, Hoffbrand AV: Long term trial with the oral iron chelator 1,2-dimethyl-3-hydroxypyrid-4-one (L1) Iron chelating and metabolic studies. Br J Haematol 76:295–300, 1990.
2. Jensen PD, Jensen IM: Desferrioxamine treatment reduces blood transfusion requirements in patients with myelodysplastic syndrome. Br J Haematol 80:121–124, 1992.
3. Vreugdenhil G, Smeets MEP, Feelders RA, van Eijk HG: Iron chelators may enhance erythropoiesis by increasing iron delivery to heamatopoietic tissue and erythropoietin response in iron loading anaemia. Acta Haematol 89:57–60, 1993.
4. Louache F, Festa U, Pelici P: Regulation of transferrin receptors in human hematopoietic cell lines. J Biol Chem 2259:11576–11582, 1984.
5. Kontoghiorges GJ, May A: Uptake and intracellular distribution of iron from transferrin and chelators in erythroid cells. Biol Metals 3:183–187, 1990.

Granulocytic Sarcoma of the Uterus Complicating Myelodysplastic Syndrome

To the Editor: Myelodysplastic syndrome (MDS) is a clonal disorder characterized by ineffective hemopoiesis and myelodysplasia [1]. The patients often present with complications of pancytopenia and the disease may terminate in acute myeloid leukemia (AML). The latter usually evolves through a gradual rise of blast count in the peripheral blood and bone marrow. Rarely, patients with MDS may develop tumorous masses of immature myeloid cells (granulocytic sarcoma), with or without concomitant systemic involvement [2]. We report a patient with MDS complicated by granulocytic sarcoma of the uterus. The latter heralded the development of frank leukemia.

A 35-year-old female presented with gum bleeding and menorrhagia in August 1993. Peripheral blood counts showed: hemoglobin 11.2 g/dL, platelets 12×10^9/L, and leukocytes 5.9×10^9/L with 55% neutrophils, 16% lymphocytes, 2% monocytes, 2% eosinophils, 4% basophils, 2% metamyelocytes, 4% myelocytes, 2% promyelocytes, and 13% Auer rod-containing blast cells. The neutrophils showed pseudo-Pelger Huet anomaly. The marrow was hypercellular with reduced megakaryocytes, megaloblastoid erythropoiesis and abnormal granulopoiesis with around 20% blast cells (500-cell differential count on separate occasions). Cytogenetic studies performed by culturing of the marrow cells revealed 45-46,XX,t(8;21)(q22;q22),del(9)(q11q22)(15). A diagnosis of refractory anemia with excess of blasts in transformation was made according to the FAB classification, although in the presence of t(8;21), this could well represent, biologically, an evolving phase of AML (M2) [3].

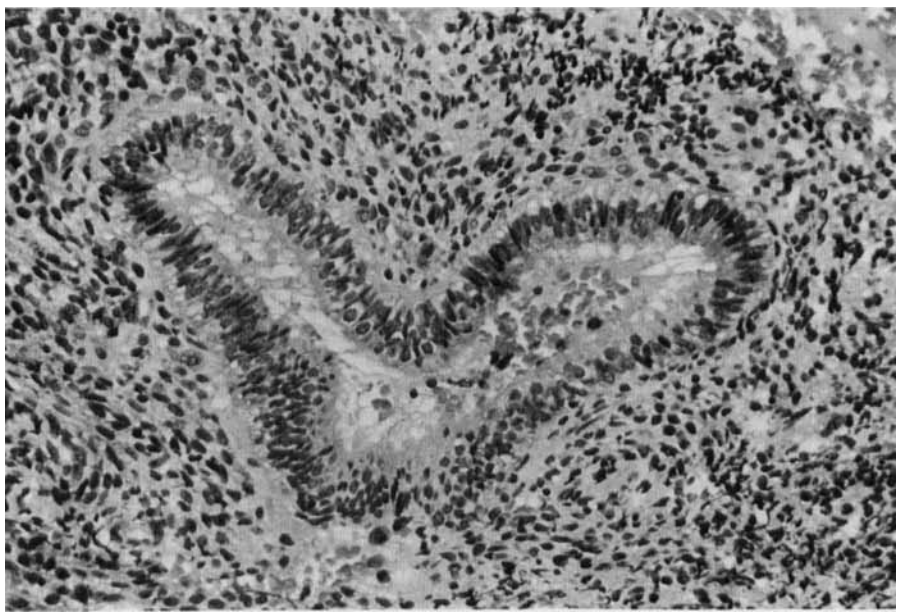

Fig. 1. Endometrial biopsy showing a compact infiltrate of myeloblasts in the endometrium (hematoxylin & eosin, ×160).

The patient was treated with daunorubicin, cytarabine, and etoposide with disappearance of blast cells from the peripheral blood and the marrow. However, her menorrhagia recurred in August 1994 and an endometrial biopsy was done, showing a diagnosis of granulocytic sarcoma (Fig. 1). At that time, peripheral blood counts were: hemoglobin 10.7 g/dL, platelets 22×10^9/L, and leukocytes 4×10^9/L with rare blast cells. Bone marrow examination showed no excess of blast cells. In September 1994, she developed a right cervical mass which was also shown to be granulocytic sarcoma on fine-needle aspiration cytology. Rapid leukemic transformation ensued after two weeks, and she was treated with intensive chemotherapy and local irradiation to the right cervical region. This was followed by persistent cytopenia, and the patient died of septicemia in November 1994.

Granulocytic sarcoma is uncommon in patients with MDS, and can occur at presentation or during blastic transformation [2]. Granulocytic sarcoma can involve virtually any anatomic site in the body, such as the lymph node, skin, nasopharynx, and gastrointestinal tract [4]. Cutaneous involvement is commonest for granulocytic sarcoma occurring in the setting of MDS [2]. However, granulocytic sarcoma involving the uterus is distinctively rare [4,5], and like granulocytic sarcoma involving other sites, the lesion is often misinterpreted as malignant lymphoma on histologic examination [5]. Our patient has an unusual presentation, with granulocytic sarcoma developing in the uterus and at a time when no evidence of leukemic transformation was observed in either the peripheral blood or bone marrow. In female patients with MDS/AML and menorrhagia, the bleeding disorder is often attributed to platelet dysfunction and thrombocytopenia. In occasional cases, however, this may be due to granulocytic sarcoma in the uterus. Detailed gynecological examination is therefore warranted, particularly when the bleeding is not explainable by the degree of thrombocytopenia.

K.F. Wong
P.H. Yu
Y.C. Chu

Department of Pathology & Medicine, Queen Elizabeth Hospital, Kowloon, Hong Kong

References

1. Bennett JM, Catovsky D, Daniel MT, Flandrin G, Galton DAG, Gralnick HR, Sultan C: Proposals for the classification of the myelodysplastic syndromes. Br J Haematol 51:189–199, 1982.
2. List AF, Gonzalez-Osete G, Kummet T, Doll DC: Granulocytic sarcoma in myelodysplastic syndromes: clinical marker of disease acceleration. Am J Med 90:274–276, 1991.
3. Kwong YL, Wong KF: Translocation (8;21)(q22;q22) and the myelodysplastic syndrome (letter). Leuk Res (in press).
4. Neiman RS, Barcos M, Berard C, Bonner H, Mann R, Rydell RE, Bennett JM: Granulocytic sarcoma: a clinicopathologic study of 61 biopsied cases. Cancer 48:1426–1437, 1981.
5. Kapadia SB, Krause JR, Kanbour AI, Hartsock RJ: Granulocytic sarcoma of the uterus. Cancer 41:687–691, 1978.

Chronic Consumption Coagulopathy and Popliteal Aneurysm

To the Editor: Chronic intravascular coagulopathy is a well-known cause of thrombocytopenia and may exceptionally complicate thoracoabdominal aneurysms [1]. We report such a coagulopathy in a patient with a popliteal aneurysm.

A 94-year-old man was referred for moderate thrombocytopenia. The patient had no medical history and no bleeding tendency. Physical examination was noticeable for the absence of arterial pulse in the left foot and a palpable left popliteal mass. Blood examination showed a moderate thrombocytopenia (60–80,000/mm^3), hemoglobin was 150 g/l, and leukocytes were 5,700/mm^3 with a normal differential count. Fibrinogen was 3 g/l, activated partial thromboplastin and prothrombin times were normal, fibrin degradation products were positive (80–160 μg/ml, N < 20 μg/ml),

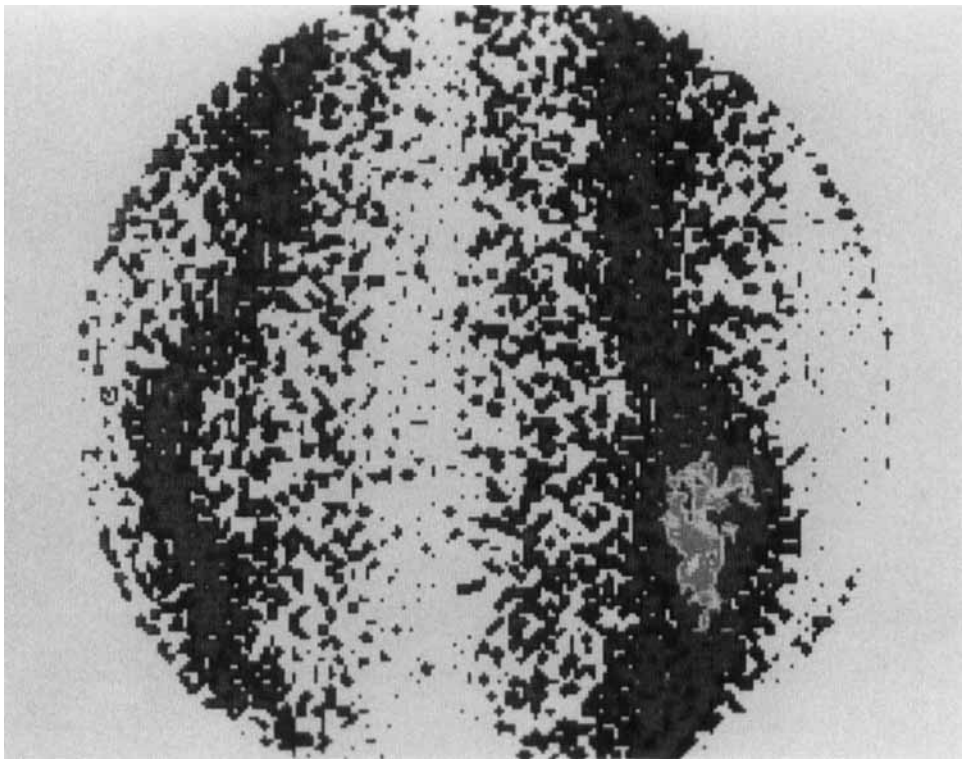

Fig. 1. Indium 111-labeled platelet scintigraphy.